

VISIT...

LANZAROTE
Caliente.COM

Lifting the Veil: A Review of the History, Clinical-
Usage and Safety of LSD, Ibogaine and MDMA in a
Psychotherapeutic Context

Christopher D. Lovett -- The University of Arizona
Program in Cognition and Neural Systems

November 1999

Introduction

To make the trivial world sublime, take half a gramme of phanerothym -- Aldous Huxley

To fathom hell or soar angelic, just take a pinch of psychedelic – Humphry Osmond (in reply)

LSD, ibogaine, MDMA and many other compounds have been used as adjuncts to psychotherapy over the last fifty years with amazing results (e.g., see Naranjo, 1973; Yensen, 1985; Grof, 1972, 1973, 1994; Walsh, 1982; Stolaroff, 1997; De-Nur, 1998; Greer & Tolbert, 1998; Metzner, 1998; Eisner, 1997; Bravo & Grob, 1996). In addition, ibogaine has been used quite effectively to interrupt and cure addictions to morphine, heroin, cocaine, amphetamine, nicotine and alcohol, with virtually no withdrawal symptoms, in both humans and animals (e.g., see Mash, *et al.*, 1998; Glick & Maisonneuve, 1998; Sharma & Bhargava, 1998; Glick, *et al.*, 1994). Though not reviewed in this paper, subanesthetic doses of ketamine have also been very successful in treating alcoholism (Krupitsky & Grinenko, 1997). Most of the ketamine research has taken place at the Leningrad Regional Center for Alcoholism and Drug Addiction Therapy in Russia, though some of the research has been conducted here in the United States at Yale University in Connecticut.

All three of these compounds, LSD, ibogaine and MDMA, have been used by psychotherapists primarily to facilitate a sense of openness and trust in the patient. Not only that, but also a feeling of acceptance as somewhat disturbing aspects of the patient's psyche are brought up and discussed during a psychotherapy session. While LSD and ibogaine are particularly intense in high dosages, MDMA seems to be much more gentle and also devoid of the distracting visual disturbances associated with the former two compounds.

The effects of each of these three compounds, while similar in being *psychedelic* ('mind-manifesting'), are different enough to belong to separate qualitative classes of compounds. Ibogaine, an alkaloid of the shrub *Tabernanthe iboga*, is traditionally used as a religious sacrament by the Mitsogho tribe of Gabon, Africa, and so would most correctly be termed an *entheogen* ('divine

generated from within'). LSD, however, being a semi-synthetic drug with no traditional usage, has most often been called a *hallucinogen* (though no true hallucinations are involved, in that the user knows what is causing the visual disturbances, i.e., the drug) or just plain *psychedelic*. While the three terms *entheogen*, *psychedelic* and *hallucinogen* occur in the literature somewhat interchangeably, MDMA and some of the other substituted amphetamines (e.g., MDA, MBDB, MDE) belong in a qualitative class of their own, the *entactogens* ('producing a touching within'), for reasons to later be explained.

Another feature these three drugs share is that they have all been scheduled by the Food and Drug Administration (FDA) and the Drug Enforcement Administration (DEA) as Schedule I substances along with heroin and cocaine. This basically means that, according to the FDA and DEA, there are no acceptable medical uses for these drugs, they have a high potential for abuse, and they are generally unsafe. Of course this also means that they are highly illegal, with stiff fines for manufacturing, distributing or possessing any quantity of these substances. This scheduling in accordance with the Controlled Substances Act of 1970 resulted largely from ad hominem propaganda put forth by the U.S. government, making research and legitimate psychotherapy with these substances nearly impossible.

It is *possible*, however, as recounted by Rick Strassman (1991), a University of New Mexico psychiatrist. This 1991 paper documents his heroic, and successful, efforts to navigate the labyrinthine rigmarole he encountered while getting approval from both the FDA and DEA to administer *N,N*-dimethyltryptamine (DMT, the active ingredient in Ayahuasca brews, and a Schedule I substance) to humans in a controlled setting. Though the process took nearly two years, Strassman's efforts finally paid off, and his published documentation of this process with every step detailed will undoubtedly prove useful to future researchers in the field.

Another seminal piece of work, which will be treasured by researchers for years to come, is contained in the two volumes published by Alexander Shulgin and his wife Ann (1992, 1997)

entitled *PIHKAL* (Phenethylamines I Have Known and Loved) and *TIHKAL* (Tryptamines I Have Known and Loved), respectively. While also including a large amount of autobiographical material, these two volumes together elucidate the syntheses, dosages, subjective effects and reports, and dangers of two-hundred and thirty-four psychoactive compounds with extraordinary clarity, detail and precision. It is likely that all of these 234 compounds, the majority of which were discovered by Shulgin himself, are potential adjuncts to psychotherapy. Needless to say, he has set the stage for decades of future research.

Given the limits of time and space (as is always the case unfortunately), this paper will be limited in scope to the examination of only three of the most popular compounds used in psychotherapy: LSD, ibogaine and MDMA. What I hope to accomplish here is to give an historical perspective, a feeling for the usefulness of each compound, and an investigation of the dangers, both psychological and physiological, involved in their usage. Hopefully, the reader will walk away more educated and better able to make an informed personal decision about the utility of these compounds based on facts, not fear.

LSD

Mainly I remember two experiences. I was alone in a timeless world with no boundaries. There was no atmosphere; there was no color, no imagery, but there may have been light. Suddenly I recognized that I was a moment in time, created by those before me and in turn the creator of others. This was my moment, and my major function had been completed. By being born, I had given meaning to my parents' existence.

Again in the void, alone without the time-space boundaries. Life reduced itself over and over again to the least common denominator. I cannot remember the logic of the experience, but I became poignantly aware that the core of life is love. At this moment I felt that I was reaching out to the world – to all people – but especially to those closest to me. I wept long for the wasted years, the search for identity in false places, the neglected opportunities, the emotional energy lost in basically meaningless pursuits. Many times, after respites, I went back, but always to variations on the same themes. The music carried and sustained me

Later, as members of my family came, there was a closeness that seemed new. That night, at home, my parents came, too. All noticed a change in me. I was radiant, and I seemed at peace, they said. I felt that way too. What has changed for me? I am living now, and being. I can take it as it comes. Some of the physical symptoms are gone. The excessive fatigue, some of the pains. I still get irritated occasionally and yell. I am still me, but more at peace. My family senses this and we are closer. All who know me well say that this has been a good experience.

[from an account by a woman in her 40s, diagnosed with terminal metastatic cancer, after an LSD session, in Bravo & Grob, 1996: pg. 338]

Albert Hofmann, a young chemist working in Switzerland for the pharmaceutical company Sandoz, first synthesized what is known as LSD in 1938. He had been working on a project preparing ergonovine and other amides of lysergic acid in the search for labor-inducing drugs which could be used by obstetricians. The 25th compound in this series of amides, created by Hofmann, was coded as LSD-25 (from the original German, **Lysergsaure-diethylamid**). After subsequent animal-testing, this compound proved to be only 70% as effective as ergonovine in causing uterine contractions and was therefore discarded (Perrine, 1996).

Five years later, prompted by a feeling that there was something more to this compound than initially discovered, Hofmann re-synthesized LSD-25. On the Friday afternoon of April 16th, 1943, after completing the synthesis Albert felt suddenly dizzy and restless so went home. When he got home, he wrote, “I sank into a not unpleasant intoxicated-like condition ... in a dreamlike state, with eyes closed ... I perceived an uninterrupted stream of fantastic pictures, extraordinary shapes with intense, kaleidoscopic play of colors. After some two hours this condition faded away,” (Hofmann, 1983: pg. 15, quoted in Perrine, 1996: pg. 260).

Reasoning that he had absorbed some of the compound trans-dermally during the synthesis, he decided to deliberately ingest some of it and note the results. On Monday, April 19th, 1943, Hofmann measured out 250 micrograms of the LSD-25 he had synthesized, dissolved it in water and drank it. Then with his assistant, he embarked on the notorious bicycle ride home during which

the effects of the drug first became noticeable, approximately 30 minutes after ingestion. Though being bombarded by distorted perceptions of light and sound and feeling frozen in time and space, Hofmann did manage to make it home where he grappled with his sanity for the next few hours.

Eventually, Sandoz pharmaceuticals released the compound for experimental use under the trade name Delysid (lysergide was the generic name). The first research with LSD-25 in the United States took place in 1949 at the Massachusetts Mental Health Center in Boston (Perrine, 1996). Delysid was available in 25-microgram tablets as well as in vials of injectable solution, and the indicated uses were to be for either psychotherapy as a *psycholytic* agent (i.e., releasing repressed material) or inducing model psychosis as a *psychotomimetic* agent (i.e., by the psychiatrist ingesting Delysid (s)he would be able to gain insight into what it was like to be psychotic or schizophrenic). Eventually it was also discovered that with very high doses of LSD (i.e., 500-1000 micrograms), patients began to have what might be called an ‘ego-dissolving’ or ‘mystical’ experience. This type of therapy, *psychedelic therapy*, became the dominant practice in the United States, most notably as practiced by Stanislav Grof and others at the Maryland Psychiatric Research Institute (Bravo & Grob, 1996).

The next stage of the LSD-research movement seems to have been a relatively prolific era. As Lester Grinspoon and James Bakalar point out, “Between 1950 and the mid-1960s there were more than a thousand clinical papers discussing 40,000 patients, several dozen books, and six international conferences on psychedelic drug therapy. It aroused the interest of many psychiatrists who were in no sense cultural rebels or especially radical in their attitudes. It was recommended for a wide variety of problems including alcoholism, obsessional neurosis, and childhood autism,” (Grinspoon & Bakalar, 1979: pg. 192). But then the doors slowly began to close on LSD-research for many reasons including the scare of genetic damage, increasing numbers of panic-attacks and emergency room admits during illicit use, and the completely irresponsible and juvenile use of this

drug by people such as Timothy Leary. In fact, this last reason was probably the most significantly detrimental to the entire field of LSD-research, and even today Mr. Leary is remembered with disdain for no less than destroying what was an intelligent and serious research-movement that had the potential for changing our entire conception and understanding of the mind. Only now in the 1990s is this movement beginning to recover, and hopefully within the next few years the amount of research being conducted will grow exponentially.

The first step in making this type of research easier to conduct, though it is not impossible at the moment, would be to de-schedule LSD, as well as certain other psychotherapeutic adjuncts, to at least a Schedule II status. Though it was initially relatively easy for physicians to obtain LSD directly from Sandoz under the Federal Food, Drug, and Cosmetics Act of 1938, new amendments to this act went into effect in 1963 severely limiting future research. Beginning in 1963 all research had to be approved by the Food and Drug Administration (FDA), the use of LSD in general medical practice was not allowed, and it could only be supplied to researchers in federal or state agencies or to those working on grants from federal or state agencies. By 1965, all LSD had to be returned to the government which was not being used in accordance with the new amendments.

Because of the widespread and careless use of LSD by much of the counter-culture of the United States, in 1966 it was made a crime in New York and California to possess LSD, and that same year Sandoz took its LSD off the market in the U.S. The final scheduling of LSD, as well as most other drugs with similar effects (e.g., mescaline and psilocybin) but also including drugs such as heroin and cocaine, came about in 1970 with the Comprehensive Drug Abuse Prevention and Control Act (Grinspoon & Bakalar, 1979). In a sub-section of this larger act called the Controlled Substances Act, there are enumerated five categories (schedules) of drugs. The most restricted class of drugs, Schedule I, includes LSD, ibogaine, MDMA and just about every other hallucinogenic drug along with the usual street drugs of abuse such as heroin, cocaine and amphetamines.

A drug classified as a Schedule I drug is considered to have no accepted medical use, lack of safety, and a high potential for abuse and/or addiction (Brody, *et al.*, 1994: pg. 875). The scheduling, though primarily determined by the Food and Drug Administration along with the Attorney General, is actually enforced by what is now known as the Drug Enforcement Administration (DEA). The penalties are extremely stiff for use, possession, distribution or manufacture of any Schedule I substance. Finally, to criminalize any newly discovered drugs, even before any of their properties have been determined, which are similar in structure or effect to those substances previously scheduled, the Controlled Substances Analog Bill of 1986 was passed. According to this bill, compounds which are structurally or functionally similar to a Schedule I drug are also considered Schedule I drugs (Strassman, 1995).

Thus, since 1970 there have been virtually no papers published on the effects of LSD or of the psychotherapeutic utility of LSD. However, even before scheduling, the legitimate use of LSD as a therapeutic agent had been stigmatized. For example, in President Lyndon Johnson's State of the Union Address in January of 1968, President Johnson referred to the "dangerous" hallucinogens such as LSD as, "these powders and pills which threaten our nation's health, vitality and self-respect," (Grob, 1998). Probably no mention was made however of the number of deaths related to alcohol, alcoholism and drunk-driving, alcohol's role in all types of crime, physical violence including domestic violence exacerbated by alcohol, the number of people in the country addicted to alcohol, etc., etc., with alcohol remaining completely unscheduled.

One of the major causes of mass hysteria during the late 1960s about the toxicity of LSD use was a paper published by Maimon Cohen and Michelle Marinello in the March 1967 issue of *Science* (Cohen & Marinello, 1967) entitled: "Chromosomal Damage in Human Leukocytes Induced by Lysergic Acid Diethylamide." This paper was widely used to further strengthen the anti-drug

propaganda being pushed on the public by the United States government, though later research conclusively revealed that LSD is not mutagenic, teratogenic or carcinogenic (Neill, 1987).

Looking in more detail at the original paper by Cohen & Marinello (1967), a glaringly obvious feature immediately jumps to the surface. In their experiments, they were adding various concentrations of LSD in solution to cultured human white blood cells (leukocytes). The concentration at which they found significant chromosomal damage was 10 micrograms/milliliter (they also tried 50 and 100 ug/ml, but this completely destroyed the cells). Now considering that human leukocytes in the human body are suspended in a total blood volume of approximately 5 liters, you find that the amount of LSD needed to be ingested for a blood-concentration of 10 ug/ml to be reached is about 50,000 micrograms or 200 times the normal 250 microgram dosage! The absurdity of these findings then becomes obvious, yet at the time of publication this scare-tactic worked very well, well enough in fact that this myth still persists and is often heard today.

A review of 68 studies conducted over the period of 1967-1971 examining whether or not LSD is carcinogenic, mutagenic or teratogenic was published in 1972 by Norman Dishotsky, *et al.* In this review, they found that the only significant genetic damage from pure LSD occurred *in vitro*, and even then only as a “result of concentrations which could not be achieved in humans with reasonable dosages,” (Dishotsky, *et al.*, 1972: pg. 439). The authors’ final conclusions were as follows: “From our own work and from a review of the literature, we believe that pure LSD ingested in moderate doses does not damage chromosomes *in vivo*, does not cause detectable genetic damage, and is not a teratogen or a carcinogen in man,” (Dishotsky, *et al.*, 1972: pg. 439). At the time of this article, however, the public mindset had already been shaped by the fear instilled by government propaganda into demonizing LSD. It is ironic that even though the review by Dishotsky, *et al.* (1972) and the paper by Cohen and Marinello (1967) had both been published in the popular yet academic

journal *Science*, the former seems to have went largely unnoticed while the latter became a significant source of hysterical propaganda.

But what of the real psychological dangers of LSD use? The perceived threat of psychological danger can at least partially be attributed to the term ‘psychotomimetic’ first ascribed to drugs such as LSD and mescaline by certain researchers (e.g., Cole & Katz, 1964). Literally meaning ‘mimicking-psychosis’, the term psychotomimetic with its implications was used because LSD was originally thought to reproduce naturally-occurring schizophrenic states in humans. But, among other reasons, the fact that a tolerance developed to LSD use led to abandoning the theory that it had any role endogenously in schizophrenia (Cole & Katz, 1964).

The most common real adverse reaction to LSD is a general panic reaction, but including in extreme cases frightening auditory and/or visual hallucinations, overwhelming anxiety, possibly aggression and violence, depression, suicidal thoughts and even paranoid delusions (Strassman, 1984). The panic reaction is usually treated in the emergency room of a hospital, in extremely severe cases with a chlorpromazine (thorazine) injection, but more often with 15-30mg of diazepam (valium). The patient is then ‘talked down’, observed for 8-10 hours and then sent home with a friend if possible. Another reported side-effect of LSD use is the occurrence of a very brief, less intense re-experiencing of the mental state one was previously in while using LSD. These are commonly called ‘flashbacks’, though they are not very common and tend to be less likely to occur over time (Hollister, 1984).

Any psychologically adverse effects of LSD, however, seem to be precipitated by non-clinical, unsupervised use for recreational purposes. Within a carefully-prepared therapeutic setting under the supervision of a competent and experienced psychotherapist, and with adequate preliminary screening of patients, the risk of adverse side-effects is negligible. To quote Rick Strassman of the University of New Mexico: “The most comprehensive reviews suggest that in well-

screened, prepared, supervised, and followed-up psychiatric patients taking pure psychedelic drugs, the incidence of serious adverse reactions is less than 1%. It is even lower in ‘normal volunteers’. Those most likely to suffer from prolonged depression, anxiety, or psychotic reactions to psychedelics are usually those with pre-existing psychiatric disorders, taking drugs of uncertain dose, nature, and quality, usually in combination with other drugs and alcohol in an uncontrolled setting,” (Strassman, 1997: pp. 154-155).

Since Sandoz pharmaceuticals began marketing its LSD to psychiatric researchers and practitioners in 1947, research and psychotherapy utilizing this substance have occurred intermittently. Originally believed to be a psychotomimetic (mimicking psychosis) compound, many researchers and therapists would ingest the drug and believe themselves to be in the same mental state as a schizophrenic patient. This myth was largely dispelled as incorrect by the late 1950s, as indicated by Manfred Bleuler’s statement at the First International Congress of Neuropsychopharmacology in 1959 that LSD, as well as mescaline, had “contributed nothing to the understanding of the pathogenesis of schizophrenia,” (Grob, 1998).

As the label of psychotomimetic was abandoned in the 1950s, so too was the term psycholytic (‘breaking-down the psyche’), though not completely. Humphry Osmond in a 1957 paper in the *Annals of the New York Academy of Sciences* proclaimed that a new word was needed for these compounds such as mescaline and LSD. Rejecting ‘psycholytic’ as too Freudian a term, in that it implied that you were breaking through to repressed memories from childhood, Osmond came up with the term *psychedelic* or ‘mind-manifesting’ (Neill, 1987). Ironically, though chosen partly because this new term would be free from other associations, in our current society today it is plagued by associations with the pop-culture of the 1960s and 1970s, most notably with the artwork, fashion and music of that micro-era. This has led to use of the slight misnomer (in that there are no true hallucinations involved) of hallucinogen, as well as the religiously-biased term *entheogen* (‘divine

generated from within’) first coined by Jonathan Ott, Carl Ruck and R. Gordon Wasson in 1973 (Wasson, *et al.*, 1986). All three of these terms are currently used somewhat interchangeably, though *entheogen* is usually reserved for ceremonial sacraments, especially when in the natural non-synthetic or non-extracted form (e.g., ibogaine).

Between the early 1950s and the mid-1960s there were literally hundreds of research papers published on LSD, so only a few of the more eminent researchers and clinicians will be mentioned here. First at the Psychiatric Research Institute in Prague, Czechoslovakia between 1956 and 1967, and then after 1967 in the United States at the Maryland Psychiatric Research Center, Stanislav Grof conducted thousands of LSD-assisted psychotherapy sessions (see e.g., Grof 1972, 1973, 1978, 1994) and is probably the most well-known and experienced of clinical researchers in this domain. After the Controlled Substances Act of 1970 the use of LSD was no longer very accessible for research, so Grof developed a regimen of specific breathing exercises which he called ‘Holotropic Breathwork’. Using these techniques, patients could achieve an altered state of consciousness and gain access to much more of their psyche while not using chemicals at all. Also prominent in the field of LSD-psychotherapy were Humphry Osmond, Abram Hoffer, Jan Bastiaans (see Snelders, 1998 and De-Nur, 1998), Betty Eisner (see Eisner, *et al.*, 1958 and Eisner, 1997), and an anonymous yet eminent psychotherapist known only as ‘Jacob’ (Stolaroff, 1997), also referred to as ‘Adam’ in Shulgin & Shulgin (1992), due to his LSD-assisted (among other psychoactive drugs) psychotherapy being forced underground with the Controlled Substances Act of 1970.

An in-depth analysis of the actual methods involved in the process of LSD-assisted psychotherapy is beyond the scope of this paper as there are many and lengthy published works specifically on this subject, and thus the original sources should be consulted for a full discussion of techniques and issues. Herein a few brief points will be discussed about some of the major methodological issues which a psychotherapist and/or client should consider. The chief concern

involved in deciding whether or not to use LSD or any other hallucinogenic compound in psychotherapy, however, should be the safety of the client. The risks and benefits, both psychological and physical, should be carefully explained and considered, and then the patient and the therapist should decide on the best course of action.

Most people within psychology are by now familiar with the terms ‘set’ and ‘setting’ insofar as how they can drastically shape the outcome of a hallucinogenic experience. *Set* refers to the mind-set of the person having the experience, their mood, personality, energy-level and general disposition on the day of the experience. *Setting* refers to the place and circumstances under which the experience takes place, whether it occurs in a psychotherapist’s office or at an all-night dance-party. Depending on these two factors, an entire range of possible experiences can occur in conjunction with the uniqueness of the individual. An interesting addition to these two classic factors is what Betty Eisner calls the *matrix* (Eisner, 1997). She explains: “Set refers to the subject; setting is the environment of the session. Matrix is the environment from which the subject comes: the environment surrounding the subject before and after the session, and the larger environment to which the subject returns,” (Eisner, 1997: pg. 214).

These three factors should be in mind when planning and preparing for any kind of chemically-assisted psychotherapy session. Screening potential clients beforehand is probably the most effective way to ensure there are no panic-reactions or bouts of severe paranoia during the session. ‘Jacob’ explains in *The Secret Chief* (Stolaroff, 1997) that the key to avoiding a troublesome session is to first develop a relationship with the patient during a few non-drug sessions, building up a mutual personal trust. He claimed to have an intuition when the right kind of bond had formed, and he felt the person would make a good candidate for chemically-facilitated psychotherapy. “Just how you know you have a good candidate is very difficult to describe. I’ve tried to relate this many times. I’ve tried to teach. It’s nothing you can teach. Only your experience will give it to you. My

intuition was the most important thing, and my stomach. My stomach would respond to something that was not right. Something they would say – or just being with them, no matter what they were saying, because I couldn't trust what they were saying as being them," (Stolaroff, 1997: pp. 50-51).

Once a candidate has been found to be suitable and a good patient-therapist relationship has been developed, the setting is the next critical factor involved in having a beneficial session. Specific styles vary widely, but usually a dimly-lit, yet pleasing, room, either at the patient's home or in the therapist's office, and the arrangement of insuring no interruptions will occur are the usual circumstances. Music is often used as a vehicle which the patient can use to 'travel' into problem areas, and also the room will often be decorated in a welcoming manner with plants, attractive pictures and a comfortable couch for the patient (Eisner, 1997). 'Jacob' would even have his patients bring specially-selected photographs of themselves and family members, arranged chronologically, to aid in evoking buried material in the psyche (Stolaroff, 1997).

As can be seen, LSD-assisted psychotherapy can involve much preparation and work before a successful session is possible. The outcome however, when the session is properly conducted and controlled, can be quite astonishing and bring about beneficial change in the patient even after years of unsuccessful traditional psychotherapy. This brief sketch hardly scratches the surface of the possibilities involved in these therapy sessions, as many transpersonal therapists emphasize Jungian archetypes, ego-dissolution, mystical experience, and of course Stanislav Grof's death-rebirth process. Hopefully, however, the general scenario and some of the issues involved have been conveyed.

Ibogaine

Now I am at the crossing of two paths inside a huge cave. Two enormous animals appear, side by side. They are of an intense pale green color. They are plant-like. They seem to be formed of some kind of cactus. Their skin is granular. Disgusting. I am impressed, but not afraid. The doctor says, "Face them." I look at them attentively. One of them has a huge head like an

elephant's – slightly funny – and from its chest hang twisted plantlike formations. When it moves, they quiver. I find it funny and disgusting.

“Imitate it. *Be that animal*,” the doctor says. I can see that I will not be able to do so. I put my legs together and try, but I do not succeed. I resist it, I don't want it, I cannot. I tremble. That is impossible. *I feel that he wants me to dance*. Did he say so, or did I imagine that? I do *not* want to dance. I don't feel like it. He insists: “Be that trembling.” I end up trying to obey. I lift my arms, surrendering to what may come. I start to tremble and I feel that my two arms are one flame, and they emit light. An energy that has come from above moves them, has put them together, and now they turn and turn as if electrified, beyond my power to stop them ... My arms burn. They are fire and continue to turn. I fall to the floor with my arms still reaching up, and gradually they begin to slow down and descend, while an infinite peace starts to invade me. It is a sweet, silent peace ...

I feel an understanding without words that I did not know before. It is consciousness. Great and deeper than ever before. I understand many ineffable things. I have not known how to love. I have lived without living. I see my little mind, when separate, as a fragment of my I AM. Understanding, consciousness – it is the same thing. There are no words, but understanding is infinite in that instant with no time.

[from an ibogaine session as described in Naranjo, 1973: pp. 206-207]

Ibogaine has a long and varied history as an entheogen (spiritual sacrament), stimulant, psychotherapeutic adjunct and anti-addictive agent. It is the principal indole alkaloid in the root-bark of *Tabernanthe iboga*, a shrub indigenous to central-west Africa and particularly Gabon. Other alkaloids include voacangine (carbomethoxy ibogaine), the positional isomer tabernanthine (13-methoxy ibogamine) and ibogamine, all of which may contribute to the overall effect of *T. iboga* (Ott, 1993). Ibogaine itself, however, has been found indigenously in other plants located throughout the world. These include *Tabernanthe orientalis* (also called *Ervatamia orientalis*) found in western Australia, *Tabernanthe pubescens* found in Zaire, and various plants in the *Tabernaemontana* genus found throughout Africa and Southeast Asia (Shulgin & Shulgin, 1997).

Used for centuries by the Mitsogho and other indigenous peoples of Gabon, Africa, the scrapings from the roots of *T. iboga* are traditionally chewed as part of the Bwiti initiation ritual (a

rite of passage from adolescence to adulthood). Called *eboka* or *mbassoka* by the Mitsogho, the scrapings of the root used in the ceremony typically contain up to about 6 grams of ibogaine (a very large dose). When used as an entheogen in this context, the initiate, or *banzi*, enters into a thirty-six hour ‘journey’ to the spirit-world, visiting his ancestors and experiencing the totality of the infinite during the ceremony. Members of the Mitsogho people also chew on the root of *T. iboga* in small quantities (containing approximately 50-150 mg of ibogaine) for its stimulant effects. This is mainly done by the men to aid in hunting vigilance and combat, and is even used as an aphrodisiac (Popik, *et al.*, 1995).

The crystalline alkaloid ibogaine was first isolated from the *Tabernanthe iboga* plant in 1901 by both J. Dybovsky & E. Landrin and A. Haller & E. Heckel, two teams of French pharmacologists. There was subsequently little interest in the compound, however, until 1939 when an extract of the roots of *Tabernanthe manii* began to be marketed in France under the trade name LambareneTM. LambareneTM, containing about 8mg of ibogaine per tablet, was used primarily for its stimulant properties by mountaineers, bicyclists and other athletes. However, in 1966 ibogaine was banned in France, in 1970 it was placed on the list of Schedule I substances within the United States and in 1989 it was banned by the International Olympic Committee (Popik & Skolnick, 1998).

Before being scheduled in the United States, ibogaine was also being used as a psychotherapeutic agent. Beginning sometime in the 1960s, the Chilean physician Claudio Naranjo began conducting psychotherapy sessions using ibogaine as a psychological catalyst. During these 4-6 hour sessions, he would orally administer a dosage of about 150-500mg (3-5mg/kg) of ibogaine to the patient. The term Naranjo used to describe ibogaine’s effects was *oneirophrenic* or ‘dream-inducing’. He described these effects as similar to those of harmaline in that the “effect on most subjects is that of eliciting vivid dreamlike sequences which may be contemplated while awake with closed eyes, without loss of contact with the environment or alterations of thinking,” while

“facilitating access to otherwise unconscious processes, feelings, or thoughts,” (Naranjo, 1973: pg. 5).

In addition to having been used as an entheogenic sacrament, a stimulant and an adjunct to psychotherapy, ibogaine has also been found to have remarkable anti-addictive properties in both humans and animals (Glick & Maisonneuve, 1998; Mash, *et al.*, 1998; Onaivi, *et al.*, 1998; Rezvani, *et al.*, 1997; Popik, *et al.*, 1994; Sharma & Bhargava, 1998; Popik, *et al.*, 1995; Popik, 1996; Glick, *et al.*, 1994). The research into this aspect of ibogaine was sparked by the serendipitous discovery of Howard Lotsof in 1962. The nineteen year-old Lotsof and a group of six or seven friends, all in various stages of addiction to either heroin or cocaine, each ingested what is thought to have been about 500 mg of ibogaine. Entering into a 36-hour waking dream-state, each of the young people had a deeply profound experience, reliving childhood memories and formative experiences they had in the past. The crucial introspective experience seemed to be a re-examining of each of their lives and gaining insight into how their addictive personality traits formed, ‘where it all went wrong’ so to say. Lotsof has compared the experience to eight to ten years of psychoanalysis compressed into a few hours (Perrine, 1996; Mash, *et al.*, 1998). He has also often compared the experience to watching a ‘slide show’ or a ‘movie run at high speed’ (Popik, 1996).

After this unexpectedly life-changing experience with ibogaine, Lotsof and five of the others who had also taken it suddenly stopped using the substance to which they had been addicted previously. This effect was permanent (though it has shown not to be this effective in all cases) and without withdrawal symptoms or subsequent cravings. After this event in 1962, Lotsof became dedicated to helping other addicts, eventually filing for and being issued five U.S. patents between 1985 and 1992 for the use of ibogaine as an anti-addictive agent. The patents were issued specifically for ibogaine being used to interrupt the addictions to: (1) morphine and heroin, (2) cocaine and

amphetamine, (3) alcohol, (4) nicotine, and also to interrupt (5) polydrug dependency (Popik, *et al.*, 1995).

To help formalize and legitimize the treatment of addicts, in 1985 Lotsof founded NDA International, based in Staten Island, New York, with the purpose of marketing ibogaine hydrochloride under the trade name Endabuse™. Each capsule of Endabuse™ contains approximately 1000mg of ibogaine, but due to scheduling by the FDA it is not yet manufactured or available within the United States. However, in recent years there have been many serious researchers interested in the efficacy and safety of ibogaine for clinical use in treating addiction. Chief among these is probably Deborah Mash at the University of Miami - School of Medicine (Mash, *et al.*, 1998) and Stanley Glick and Isabelle Maisonneuve at Albany Medical College (SUNY) in upstate New York (e.g., Glick & Maisonneuve, 1998; Glick, *et al.*, 1998; Glick, *et al.*, 1994; Wei, *et al.*, 1998; Molinari, *et al.*, 1996).

Deborah Mash, along with others, began conducting FDA-approved Phase I Pharmacokinetic and Safety Trials in male humans in 1995. This following previous trials using African Green monkeys in which no evidence of neurotoxicity was found (Popik, *et al.*, 1995). Unfortunately, the National Institute on Drug Abuse (NIDA) suspiciously withdrew funding for these human safety trials, and therefore the study was never completed. One may speculate that the NIDA foresaw an end to drug addiction which would effectively lead to the end of their organization along with the many jobs and huge sums of money involved. Ironically, if this is true, the NIDA would like to avoid ending drug addiction, contradicting the purpose for which it was created, for practical and economic reasons. According to Howard Lotsof (1999), however, “Deborah Mash and the University of Miami and NDA/myself have been locked in litigation for two years now concerning their failure to complete the FDA-approved Phase I study and concurrently developing competing products (Bioactive Tricyclic Ibogaine Analogs).”

On a positive note, Deborah Mash and others have been treating addicts with ibogaine since the mid-1990s, officially outside of the United States on the island of St. Kitts, through Healing Visions: Institute for Addiction Recovery, Ltd. Also, in the late-1980s and early-1990s, Howard Lotsof was involved in treating addicts in the Netherlands in conjunction with the International Coalition for Addict Self-Help (ICASH) and the Dutch Addict Self-Help (DASH) groups. Regarding the subjective reports of many addicts under the influence of ibogaine re-living past-events, a similar finding has recently been found in rats (Popik, 1996). This interesting study found that ibogaine, when given in a 2.5 mg/kg dosage, facilitated memory retrieval in the rats as measured by the Morris maze spatial navigation task. This finding may prove to be key in understanding the anti-addictive properties of ibogaine on a non-physiological level.

Recently a new ibogaine congener, 18-methoxycoronaridine (MEC), has been found to also be an effective anti-addictive agent (Glick, *et al.*, 1996; Rho & Glick, 1998; Glick, *et al.*, 1998; Rezvani, *et al.*, 1997). Whereas ibogaine itself has been found to cause the degeneration of Purkinje cells in rat cerebellar vermis at the stupendously high dosage of 100 mg/kg (Popik, *et al.*, 1995) (normal human dosage is about 10-15 mg/kg for anti-addictive effects), MEC showed no such neurotoxicity even at 100 mg/kg (Glick, *et al.*, 1996). Another purported benefit of MEC is that it involves no hallucinogenic effects as are seen with ibogaine, although as Lotsof (1999) states: “Work on MEC remains pretty confidential and it probably has not yet been determined if it has hallucinogenic properties or not.”

MDMA

Janice, her son, and I, all three of us, took 120 milligrams of MDMA in the early afternoon, and the son went off by himself. At about the half-hour point, the usual “awareness” time, Janice gave no indication of effects, nor were there any changes at the 40 minute nor 50 minute point. A few off-hand comments were offered.

“My throat is dry.”

“I’ll get you a glass of water.” Which I did. It did no good.

“I’m having trouble breathing.”

“So, breathe as best you can.” I noticed by the reflection in the window where we were, at the back of the house, that she had no difficulty breathing when I wasn’t watching her.

We walked up the hill, to an area I had leased out to the condominium builders on the neighboring land for storage of lumber. There were several ‘no smoking’ signs around as fire warnings.

“Do you think I smoke too much?”

“Do *you* think you smoke too much?”

“I don’t think so.”

“Then the answer is: probably not.”

It was now an hour into the experiment, and still no acknowledgment of any activity from the MDMA. Then, came the unexpected question, the “off the wall” question.

“Is it all right to be alive?”

“You bet your sweet ass it’s all right to be alive! It’s grace to be alive!”

That was it. She plunged into the MDMA state, and started running down the hill, calling out that it was all right to be alive. All the greens became living greens and all the sticks and stones became vital sticks and stones. I caught up with her and her face was radiant. She told me some of her personal history which she knew well, and which I knew well, but with which she had never come to peace.

She had come into the world by an unexpected Caesarean section and her mother had died during the delivery. And for fifty years she had lived in the guilt of having had her life given her at the cost of her mother’s life. She had been in therapy with her family physician for about three years, largely addressing this problem, and apparently what she needed was the acknowledgment that it was all right to be alive.

I didn’t hear from her for a couple of months. When she did call, she volunteered that she still felt very much at peace, and had discontinued her therapy.

[from Shulgin & Shulgin, 1992: pp. 71-72]

3,4-Methylenedioxymethamphetamine (MDMA) is yet another compound with a long and circuitous history as a psychotherapeutic adjunct. MDMA is the *N*-methylated congener of 3,4-methylenedioxyamphetamine (MDA), a popular street-drug and psychotherapeutic agent of the late

1960s. MDA, also known as the “Hug Drug” or “Mellow Drug of America” within the 1960s counter-culture, has been described as enhancing feelings of well-being, empathy, insight and self-awareness while decreasing levels of anxiety and emotional inhibition (Perrine, 1996). For these effects, MDA has been used during psychotherapy to help facilitate the expression and integration of meaningful, disturbing emotional content that a patient might be chronically experiencing and dwelling upon (e.g., Naranjo, 1973; Stolaroff, 1997; Yensen, *et al.*, 1976). Andrew Weil has also reported an increase in muscular coordination with MDA: “I have also tried things like rock climbing and swimming after taking MDA and again find that my body works in a more coordinated, smoother fashion and that I can do things with it that I usually cannot,” (Weil, 1976: pg. 335). Also, in contrast to LSD which seems to ‘demand’ inner exploration, MDA has been reported to be more gentle in ‘inviting’ introspection (Yensen, *et al.*, 1976).

MDMA, which didn’t become very well-known until the early-1970s, was actually first synthesized some time during 1912. The pharmaceutical firm E. Merck applied for a patent on the compound on December 24th, 1912 and was issued that patent on May 16th, 1914 (though the patent has since expired making the compound public domain) in Darmstadt, Germany (Shulgin, 1986). There is a common myth sometimes heard that MDMA was first patented as an appetite suppressant, but this rumor is completely false. MDMA was synthesized during an investigation of *precursors* to be used in making other compounds (Cohen, 1998).

MDMA seems to have remained in relative obscurity until the 1950s, when it was investigated by the U.S. Army as Edgewood Arsenal experimental agent 1475 (EA-1475), most likely as a potential ‘truth-serum’ (Cohen, 1998). There is also a reference to its synthesis being investigated in Poland in 1960 (Shulgin, 1986), and Alexander Shulgin himself did synthesize it in 1965 while working at Dow Pharmaceuticals, but he had never tried consuming it (Shulgin & Shulgin, 1992).

It wasn't until 1967 or 1968 when Alexander Shulgin met Merrie Kleinman, a young chemistry graduate student at the University of California – San Francisco, that his interest in MDMA was renewed. She told him that she had synthesized some of the *N*-methylated MDA, and with two close friends had tried it (Perrine, 1996). She and her friends each having ingested 100 mg of MDMA, Merrie said of the experience only that it was quite emotional but overall left them with a good feeling. Shulgin then synthesized it again himself and ingested some, finding it “unlike anything I had taken before,” (Shulgin & Shulgin, 1992).

Alexander Shulgin's synthesis of MDMA at the end of the 1960s marks the introduction of this compound into the circles of psychotherapists and researchers he knew interested in exploring the hidden recesses of the mind. This time marked the introduction of MDMA onto the street as well, though it did not gain its massive popularity of today until perhaps 1980. By the mid-1970s however, MDMA-assisted psychotherapy had become very popular, as it had not yet been scheduled (unlike MDA, which was included in the Controlled Substances Act of 1970) and had produced amazing results in therapy sessions (Cohen, 1998). The primary benefits with which MDMA is attributed include: having little psychological risk (compared to LSD), effects lasting only 3 to 5 hours (again, compared to LSD's 6-10 hours), enhanced bonding and communication between the therapist and the patient or between the two partners of a couple, facilitation of the expression of emotions and opinions, and little or no perceptual or cognitive distortions or loss of ego control (again, in contrast to LSD) (Greer, 1985; see also Greer and Tolbert, 1986, 1998; Liester, *et al.*, 1992; Nichols, 1986; Stolaroff, 1997; Downing, 1986).

The effects and chemistry of MDMA (and MDA) are in fact so different than typical hallucinogens that they are deserving of their own name as a qualitative class of compounds. The case for this is quite thoroughly and convincingly made by David Nichols (1986), who gives several arguments for the new class name of *entactogens*. “First of all, MDA itself [and MDMA] is really a

unique compound among the so-called hallucinogens. It is not generally used for its hallucinogenic effect, but rather for its affect-enhancing qualities,” (Nichols, 1986: pg. 306). Secondly, all other hallucinogenic amphetamines (e.g., the substituted amphetamine DOM) are active only in their *levo*-, or R(-), optical isomer while MDMA is active in its *dextro*-, or S(+), optical isomer, and MDA is active in both forms though with differing effects in each. Thus, MDMA and MDA cannot be acting at exactly the same site of action in the brain as other hallucinogenic amphetamines, because a “structurally well-defined receptor does not suddenly *decide* to accept the *dextro*- isomer for one compound, when for all other members of a drug series [(i.e., the substituted amphetamines)] it prefers the *levo*- isomer,” (Nichols, 1986: pg. 307). It is energetically impossible due to bonding constraints at the molecular level.

Finally, it is well-known that in all other hallucinogenic substituted amphetamines, if you change the alpha-methyl group to an alpha-ethyl group, you lose all hallucinogenic activity (most likely because the molecule no longer fits the receptor). To test this out in MDMA, Nichols and his research team substituted an alpha-ethyl group for the alpha-methyl group on MDMA and the resulting compound was *N*-methyl-1-(1,3-benzodioxol-5-yl)-2-butanamine, or MBDB. If this compound followed the trend of all other hallucinogenic substituted amphetamines, then it would be expected to lose all hallucinogenic activity. When tested in both humans and rats, however, this was not the case. MBDB did have effects in humans and rats similar to MDMA, though less potent, and also the active form was the *dextro*- isomer as with MDA and MDMA (Nichols, 1986).

Because of these three key pieces of evidence, Nichols and his team felt a new term was needed for this chemical class of compounds. Rejecting *empathogen* as too limiting and also suggesting deleterious effects (i.e., “pathogen”), they came up with the term *entactogen*. In Nichols’ words: “It seemed that the effect of these drugs was to enable the therapist – or patient – to reach inside and deal with painful emotional issues that are not ordinarily accessible. Just as the word ‘tact’ has the

connotation of communicating information in a sensitive and careful way, so as to avoid offense, it seemed that the Latin root of this word, *tactus*, would be appropriate as part of the term. Addition of the Greek roots *en* (within or inside) and *gen* (to produce) created the term ‘entactogen’, having the connotation of *producing a touching within*,” (Nichols, 1986: pg. 308).

By the mid-1980s, while still prospering as a psychotherapeutic agent, MDMA had caught-on in the dance-club scene, especially in Texas. “What had been a low-level production from chemists in the Boston area was superseded when one of their Southwest distributors started his own operation in Texas, and renamed the drug ‘Ecstasy,’” (Perrine, 1996: pg. 304). It became so available in Texas bars and nightclubs at one point, that supposedly some establishments even allowed purchases by credit-card and sometimes even handed it out as a free promotional item (Cohen, 1998). 1984, however, was the beginning of the end when Lloyd Bentsen, a Texas Senator, made the first request to the DEA to schedule MDMA.

This action sparked off two years of heated debate about whether or not to place MDMA in Schedule I, with both sides emphatic in their beliefs. A crucial piece of evidence used by the DEA, though resting on the misinformation of MDMA being synonymous with MDA, was a paper published in *Science* in 1985 by George Ricaurte and his team (i.e., Ricaurte, *et al.*, 1985). The data of this paper indicated that a 10 mg/kg dosage of MDA (equivalent to a 500-1000 mg dose in humans) caused a reduction in serotonin levels in the rat forebrain. Although, ironically, one of the conclusions made by the authors was that “the doses of MDA typically ingested by humans may not be sufficiently high to induce serotonin neurotoxicity, unless humans are more sensitive than rats to the toxic effects of MDA,” (Ricaurte, *et al.*, 1985: pg. 988). After even more court hearings, MDMA was eventually placed in Schedule I, temporarily in July of 1985, and permanently on November 13, 1986 (Cohen, 1998). On December 22nd, 1987, it was temporarily placed in Schedule III (accepted medical use but moderate potential for abuse and physical and/or psychological dependency) due to

subsequent court hearings and a decision that there were indeed acceptable medical uses for MDMA, but unfortunately this decision was reversed and on March 23rd, 1988 it was placed back in Schedule I (Liestner, *et al.*, 1992).

Not much changed in any positive way with the scheduling of MDMA, in my opinion. Effectively, these were the real consequences: the price on the street soared to \$20-\$25 per tablet or capsule; the quality, purity and exact amount of MDMA in each capsule or tablet became even more uncertain; previously legal psychotherapy was forced underground; the criminal element of the ‘black market’ brought a new factor of potential violence, inherent in such illegal dealings involving large sums of money, to all levels of MDMA manufacture, distribution and, now, smuggling of both product and precursors; and lastly, the consumption of MDMA seems to have went up, not down, since 1985 with the massive popularization of the drug (now known as ‘E’, ‘X’ and ‘XTC’) in the underground dance-club, or ‘rave’, scene which emerged in the United States and England in the mid-1980s.

Despite the usual governmental propaganda, perhaps we should examine the real risks involved in MDMA use. It seems, at least psychologically, extremely safe especially when compared to most legally-prescribed drugs: “... in 1986, J. Newmeyer conducted a review showing that adverse reactions to MDMA averaged fewer than 20 per year from all emergency rooms nationwide, despite literally millions of individuals using the drug,” (Perrine, 1996: pg. 304). A very real risk, however, which is associated with MDMA use in the rave-scene, is the danger of heat-exhaustion when dancing for hours on end without drinking any water or juice, especially when the venue is indoors and over-crowded. Heat-exhaustion and hyperthermia have caused many casualties and even fatalities associated with, though not caused by, MDMA use.

The neurotoxicity of MDMA however, remains somewhat unclear, though if there is any at all it seems to be no more than that caused by such prescription drugs as Fenfluramine, a Schedule

IV drug used to treat obesity (e.g., see Frederick, *et al.*, 1998 and McCann & Ricaurte, 1995). The glaringly obvious difference which automatically appears here, however, is that while Fenfluramine (which has been used by over 50 million patients) is taken **daily** for sometimes many consecutive months, a dose of MDMA used in psychotherapy may be given only once or twice a year, if that. Even McCann and Ricaurte (proponents of MDMA neurotoxicity) have said, “Although it is not clear why the potential for serotonin neurotoxicity in humans receiving Fenfluramine has not received much attention, it may be, in part, related to its use in a more socially acceptable therapeutic setting,” (McCann & Ricaurte, 1995).

Regarding the actual neurotoxicity caused by both MDMA and Fenfluramine in animals (not humans), they have both been found in monkeys and rats to deplete serotonin levels as measured by reduced levels of 5-hydroxyindoleacetic acid (5-HIAA), a metabolite of serotonin, in the cerebral spinal fluid (CSF) (e.g., Ricaurte, *et al.*, 1988a; Steele, *et al.*, 1994; Hegadoren, *et al.*, 1995; Fischer, *et al.*, 1995; McCann, *et al.*, 1996), reduced levels of serotonin as measured directly (e.g., Ricaurte, *et al.*, 1988b), and increased locomotor activity (Nagilla, *et al.*, 1998). The reduced serotonin levels seem to be due to changes in serotonergic neurons, specifically at pre-synaptic serotonin uptake sites (Sprague, *et al.*, 1998). Even though these studies with high dosages and/or repeated administrations of MDMA have shown some serotonin depletion in animals, a 1993 primate study was conducted by George Ricaurte that unfortunately was never published indicating that a single experimental/therapeutic dose MDMA was probably not neurotoxic at all. In an October, 1999 letter to Neuropsychopharmacology, Franz X. Vollenweider, *et al.*, state: “Finally, 2.5 mg/kg MDMA given p.o. once every 2 weeks for 4 months did not alter 5-HT and 5-HIAA brain content in squirrel monkeys (Ricaurte 1993, pers. comm. to the Swiss Federal Ethical Committee),” (Vollenweider, *et al.*, 1999: pg. 599).

There are several problems with neurotoxicological studies being generalized to humans occasionally using a therapeutic-dose of MDMA in a clinical setting. First, the dosages used in rats and monkeys have been anywhere from 20-80 mg/kg, sometimes administered subcutaneously, intramuscularly or intraperitoneally (i.e., without a ‘first pass’ through the stomach and liver) (Hegadoren, *et al.*, 1999). Also, 20-80 mg/kg in a 50-100 kg (110-220 lb) human equals 1000-80,000 mg of MDMA consumed, and in these studies this dosage was sometimes given repeatedly in one day or over a few days to elicit the reported neurotoxicity. These dosages are quite high, to say the least, given the normal therapeutic dose of 50-250 mg. Another problem, perhaps supporting the strongest case against the neurotoxicity data, is that generalizing findings across species is by itself inconclusive. Dan Perrine (1996) points out that “while serotonin neurodamage in monkeys from MDMA appears to be permanent, in rats function seems slowly to return to normal,” (pg. 305). So presently, the debate still rages on as to whether MDMA cause neurotoxicity or not. It does seem tragic however, that because of its Schedule I status, the research which needs to be done on MDMA to show its probable harmlessness and already-proven clinical efficacy has been severely hampered while drugs such as Fenfluramine continue to be prescribed on a regular basis.

Conclusions

There I was, poised in space, a disembodied eye, invisible, incorporeal, seeing but not seen

-- R. Gordon Wasson (1957)

Despite the perseverance of the United States government in attempting to police the insides of people’s bodies, even to police people’s minds, research, psychotherapy and recreational use of many Schedule I substances flourishes in today’s society. Renewed research interest in LSD, but most notably in ibogaine and MDMA, during the 1990s can be witnessed in the abundance of recent research papers. The first meeting devoted to the topic of ibogaine at a U.S. academic medical center

will be held this November 5th & 6th, at New York University's School of Medicine (for info., see <http://www.med.nyu.edu/Psych/ibogaineconf/>).

Additionally, such organizations as the Multidisciplinary Association for Psychedelic Studies (MAPS) and the Heffter Research Institute have emerged with the sole purpose of funding, facilitating and publishing research and information related to psychedelics (e.g., see <http://www.maps.org> and <http://www.heffter.org>, respectively). In fact, MAPS will be sponsoring a scientific conference on clinical research with MDMA, August 30th – September 1st, 1999, in Israel. As can be seen, the resurgent interest and motivation in researching substances such as LSD, ibogaine and MDMA is growing everyday.

Perhaps one day we will live in a world where addiction and emotional misery are largely things of the past, as today are Polio and Tuberculosis. It seems ironic, however, that in today's society anyone over 18 years of age (and most people younger than 18 as well) can buy tobacco almost anywhere, with only a small warning label required on the package. Tobacco, highly addictive, with no therapeutic use, and proven to cause many health-related problems and many instances of death, remains completely legal and unscheduled. Substances such as MDMA and LSD on the other hand, non-addictive, highly beneficial to mankind when used therapeutically, and with no undisputedly long-term toxic effects, especially at therapeutic dosages, are among some of the most highly restricted and controlled substances on the planet.

I don't get it, do you?

References

- Abraham, H.D. & Aldridge, A.M. (1993). Adverse consequences of lysergic acid diethylamide. *Addiction*, **88**: 1327-1334.
- Bravo, G. & Grob, C. (1996). Psychedelic psychotherapy. In B.W. Scotton, A.B. Chinen & J.R. Battista (Eds.), *Textbook of transpersonal psychiatry and psychology* (pp. 335-343). New York: Basic Books.
- Brody, T.M., Larner, J., Minneman, K.P. & Neu, H.C. (1994). *Human pharmacology*, 2nd ed. St. Louis, MO: Mosby-Year Book, Inc.
- Cohen, M.M. & Marinello, M.J. (1967). Chromosomal damage in human leukocytes induced by lysergic acid diethylamide. *Science*, **155**: 1417-1419.
- Cohen, R.S. (1998). *The love drug: Marching to the beat of ecstasy*. Binghamton, NY: The Haworth Medical Press.
- Cole, J.O. & Katz, M.M. (1964). The psychotomimetic drugs. *JAMA*, **187**(10): 758-761.
- Cooper, J.R., Bloom, F.E. & Roth, R.H. (1996). *The biochemical basis of neuropharmacology*, 7th ed. NY: Oxford University Press, Inc.
- Cosgrove, J.W. & Brown, I.R. (1984). Effect of intravenous administration of D-lysergic acid diethylamide on initiation of protein synthesis in a cell-free system derived from brain. *Journal of Neurochemistry*, **42**: 1420-1426.
- De-Nur, Y. [Ka-Tzetnik 135633] (1998). *Shivitti: A vision*. Translated from the Hebrew by Eliyah Nike De-Nur and Lisa Herman. Nevada City, CA: Gateways/IDHHB Publishers.
- De Souza, I., Kelly, J.P., Harkin, A.J. & Leonard, B.E. (1997). An appraisal of the pharmacological and toxicological effects of a single oral administration of 3,4-methylenedioxymethamphetamine (MDMA) in the rat. *Pharmacology & Toxicology*, **80**: 207-210.
- Dishotsky, N.I., Loughman, W.D., Mogar, R.E. & Lipscomb, W.R. (1971). LSD and genetic damage. *Science*, **172**: 431-440.
- Downing, J. (1986). The psychological and physiological effects of MDMA on normal volunteers. *Journal of Psychoactive Drugs*, **18**(4): 335-340.
- Eisner, B. (1997). Set, setting, and matrix. *Journal of Psychoactive Drugs*, **29**(2): 213-216.
- Eisner, B. & Cohen, S. (1958). Psychotherapy with lysergic acid diethylamide. *Journal of Nervous and Mental Disease*, **127**: 528-539.

- Fischer, C., Hatzidimitriou, G., Wlos, J., Katz, J. & Ricaurte, G. (1995). Reorganization of ascending 5-HT axon projections in animals previously exposed to the recreational drug ($\pm$)3,4-methylenedioxymethamphetamine (MDMA, "Ecstasy"). *The Journal of Neuroscience*, **15**(8): 5476-5485.
- Frederick, D.L., Ali, S.F., Gillam, M.P., Gossett, J., Slikker, W. Jr. & Paule, M.G. (1998). Acute effects of dexfenfluramine (d-FEN) and methylenedioxymethamphetamine (MDMA) before and after short-course, high-dose treatment. In S.F. Ali (Ed.), *The neurochemistry of drugs of abuse: cocaine, ibogaine, and substituted amphetamines* (pp. 183-190). Annals of the New York Academy of Sciences, Volume **844**.
- Glick, S.D., Kuehne, M.E., Raucci, J., Wilson, T.E., Larson, D., Keller, R.W. Jr. & Carlson, J.N. (1994). Effects of *iboga* alkaloids on morphine and cocaine self-administration in rats: relationship to tremorigenic effects and to effects on dopamine release in nucleus accumbens and striatum. *Brain Research*, **657**: 14-22.
- Glick, S.D., Kuehne, M.E., Maisonneuve, I.M., Bandarage, U.K. & Molinari, H.H. (1996). 18-methoxycoronaridine, a non-toxic *iboga* alkaloid congener: effects on morphine and cocaine self-administration and on mesolimbic dopamine release in rats. *Brain Research*, **719**: 29-35.
- Glick, S.D. & Maisonneuve, I.M. (1998). Mechanisms of antiaddictive actions of ibogaine. In S.F. Ali (Ed.), *The neurochemistry of drugs of abuse: cocaine, ibogaine, and substituted amphetamines* (pp. 214-226). Annals of the New York Academy of Sciences, Volume **844**.
- Glick, S.D., Maisonneuve, I.M., Visker, K.E., Fritz, K.A., Bandarage, U.K. & Kuehne, M.E. (1998). 18-methoxycoronaridine attenuates nicotine-induced dopamine release and nicotine preferences in rats. *Psychopharmacology*, **139**: 274-280.
- Greer, G. (1985). Using MDMA in psychotherapy. *Advances*, **2**(2): 57-59.
- Greer, G. & Tolbert, R. (1986). Subjective reports of the effects of MDMA in a clinical setting. *Journal of Psychoactive Drugs*, **18**(4): 319-327.
- Greer, G.R. & Tolbert, R. (1998). A method of conducting therapeutic sessions with MDMA. *Journal of Psychoactive Drugs*, **30**(4): 371-379.
- Grinspoon, L. & Bakalar, J.B. (1979). *Psychedelic drugs reconsidered*. NY: Basic Books, Inc.
- Grob, C.S. (1998). Psychiatric research with hallucinogens: What have we learned? *The Heffter Review of Psychedelic Research*, **1**: 8-20.
- Grob, C.S., Greer, G.R. & Mangini, M. (1998). Hallucinogens at the turn of the century: An introduction. *Journal of Psychoactive Drugs*, **30**(4): 315-319.
- Grof, S. (1972). Varieties of transpersonal experiences: Observations from LSD psychotherapy. *Journal of Transpersonal Psychology*, **4**(1): 45-80.
- Grof, S. (1973). Theoretical and empirical basis of transpersonal psychology and psychotherapy: Observations from LSD research. *Journal of Transpersonal Psychology*, **5**(1): 15-53.

- Grof, S. (1988). *The adventure of self-discovery: Dimensions of consciousness and new perspectives in psychotherapy and inner exploration*. Albany, NY: State University of New York Press.
- Grof, S. (1994). *LSD psychotherapy*, 2nd ed. Alameda, CA: Hunter House Inc.
- Hegadoren, K.M., Martin-Iverson, M.T. & Baker, G.B. (1995). Comparative behavioural and neurochemical studies with a psychomotor stimulant, an hallucinogen and 3,4-methylenedioxy analogues of amphetamine. *Psychopharmacology*, **118**: 295-304.
- Hegadoren, K.M., Baker, G.B. & Bourin, M. (1999). 3,4-Methylenedioxy analogues of amphetamine: Defining the risks to humans. *Neuroscience and Biobehavioral Reviews*, **23**: 539-553.
- Hofmann, A. (1983). *LSD, my problem child*. Los Angeles, CA: J.P. Tarcher (St. Martin's Press).
- Hollister, L.E. (1984). Effects of hallucinogens in humans. In B.L. Jacobs (Ed.), *Hallucinogens: Neurochemical, behavioral, and clinical perspectives* (pp. 19-33). NY: Raven Press.
- Krupitsky, E.M. & Grinenko, A.Y. (1997). Ketamine psychedelic therapy (KPT): A review of the results of ten years of research. *Journal of Psychoactive Drugs*, **29**(2): 165-183.
- Liester, M.B., Grob, C.S., Bravo, G.L. & Walsh, R.N. (1992). Phenomenology and sequelae of 3,4-methylenedioxymethamphetamine use. *The Journal of Nervous and Mental Disease*, **180**(6): 345-356.
- Lotsof, H. (1999). [Personal communication].
- Mangini, M. (1998). Treatment of alcoholism using psychedelic drugs: A review of the program of research. *Journal of Psychoactive Drugs*, **30**(4): 381-417.
- Mash, D.C., Kovera, C.A., Buck, B.E., Norenberg, M.D., Shapshak, P., Hearn, W.L. & Sanchez-Ramos, J. (1998). Medication development of ibogaine as a pharmacotherapy for drug dependence. In S.F. Ali (Ed.), *The neurochemistry of drugs of abuse: cocaine, ibogaine, and substituted amphetamines* (pp. 274-292). Annals of the New York Academy of Sciences, Volume **844**.
- McCann, U.D. & Ricaurte, G.A. (1995). On the neurotoxicity of MDMA and related amphetamine derivatives. *Journal of Clinical Psychopharmacology*, **15**(4): 295-296.
- McCann, U.D., Slate, S.O. & Ricaurte, G.A. (1996). Adverse reactions with 3,4-methylenedioxy-methamphetamine (MDMA; 'Ecstasy'). *Drug Safety*, **15**(2): 107-115.
- McCann, U.D., Szabo, Z., Scheffel, U., Dannals, R.F. & Ricaurte, G.A. (1998). Positron emission tomographic evidence of toxic effect of MDMA ("Ecstasy") on brain serotonin neurons in human beings. *The Lancet*, **352**: 1433-1437.
- McGlothlin, W., Cohen, S. & McGlothlin, M.S. (1970). Long lasting effects of LSD on normals. *Journal of Psychedelic Drugs*, **3**(1): 20-31.

- Metzger, R.R., Hanson, G.R., Gibb, J.W. & Fleckenstein, A.E. (1998). 3,4-Methylenedioxymethamphetamine-induced acute changes in dopamine transporter function. *European Journal of Pharmacology*, **349**: 205-210.
- Metzner, R. (1998). Hallucinogenic drugs and plants in psychotherapy and shamanism. *Journal of Psychoactive Drugs*, **30**(4): 333-341.
- Molinari, H.H., Maisonneuve, I.M. & Glick, S.D. (1996). Ibogaine neurotoxicity: a re-evaluation. *Brain Research*, **737**: 255-262.
- Nagilla, R., Newland, M.C., Snyder, J. & Bronson, M.E. (1998). Effect of once weekly treatment with 3,4-methylenedioxymethamphetamine on schedule-controlled behavior in rats. *European Journal of Pharmacology*, **358**: 1-8.
- Naranjo, C. (1973). *The healing journey: New approaches to consciousness*. NY: Pantheon Books (A division of Random House).
- Neill, J.R. (1987). "More than medical significance": LSD and American psychiatry 1953 to 1966. *Journal of Psychoactive Drugs*, **19**(1): 39-45.
- Nichols, D.E. (1986). Differences between the mechanism of action of MDMA, MBDB, and the classic hallucinogens. Identification of a new therapeutic class: Entactogens. *Journal of Psychoactive Drugs*, **18**(4): 305-313.
- Onaivi, E.S., Ali, S.F. & Chakrabarti, A. (1998). *In Vivo* ibogaine blockade and *In Vitro* PKC action of cocaine. In S.F. Ali (Ed.), *The neurochemistry of drugs of abuse: cocaine, ibogaine, and substituted amphetamines* (pp. 227-244). Annals of the New York Academy of Sciences, Volume **844**.
- Ott, J. (1993). *Pharmactheon: Entheogenic drugs, their plant sources and history*. Kennewick, WA: Natural Products Co.
- Perrine, D.M. (1996). *The chemistry of mind-altering drugs: History, pharmacology, and cultural context*. Washington, DC: American Chemical Society.
- Popik, P. (1996). Facilitation of memory retrieval by the "anti-addictive" alkaloid, ibogaine. *Life Sciences*, **59**(24): PL379-PL385.
- Popik, P., Layer, R.T. & Skolnick, P. (1994). The putative anti-addictive drug ibogaine is a competitive inhibitor of [³H]MK-801 binding to the NMDA receptor complex. *Psychopharmacology*, **114**: 672-674.
- Popik, P., Layer, R.T. & Skolnick, P. (1995). 100 years of ibogaine: Neurochemical and pharmacological actions of a putative anti-addictive drug. *Pharmacological Reviews*, **47**(2): 235-253.
- Popik, P. & Skolnick, P. (1998). Pharmacology of ibogaine and ibogaine-related alkaloids. In G.A.

- Cordell (Ed.), *The alkaloids: Chemistry and biology (Vol. 52)* (pp. 197-231). San Diego, CA: The Academic Press.
- Rezvani, A.H., Overstreet, D.H., Yang, Y., Maisonneuve, I.M., Bandarage, U.K., Kuehne, M.E. & Glick, S.D. (1997). Attenuation of alcohol consumption by a novel nontoxic ibogaine analogue (18-methoxycoronaridine) in alcohol-preferring rats. *Pharmacology Biochemistry and Behavior*, **58**(2): 615-619.
- Rho, B. & Glick, S.D. (1998). Effects of 18-methoxycoronaridine on acute signs of morphine withdrawal in rats. *NeuroReport*, **9**(7): 1283-1285.
- Ricuarte, G.A., Bryan, G., Strauss, L., Seiden, L. & Schuster, L. (1985). Hallucinogenic amphetamine selectively destroys brain serotonin nerve terminals. *Science*, **229**: 986-988.
- Ricaurte, G.A., DeLanney, L.E., Wiener, S.G., Irwin, I. & Langston, J.W. (1988a). 5-Hydroxyindole-acetic acid in cerebrospinal fluid reflects serotonergic damage induced by 3,4-methylenedioxymethamphetamine in CNS of non-human primates. *Brain Research*, **474**: 359-363.
- Ricaurte, G.A., DeLanney, L.E., Irwin, I. & Langston, J.W. (1988b). Toxic effects of MDMA on central serotonergic neurons in the primate: importance of route and frequency of drug administration. *Brain Research*, **446**: 165-168.
- Schechter, M.D. (1998). 'Candyflipping': Synergistic discriminative effect of LSD and MDMA. *European Journal of Pharmacology*, **341**: 131-134.
- Scheffel, U., Szabo, Z., Mathews, W.B., Finley, P.A., Dannals, R.F., Ravert, H.T., Szabo, K., Yuan, J. & Ricaurte, G.A. (1998). In vivo detection of short- and long-term MDMA neurotoxicity – A positron emission tomography study in the living baboon brain. *Synapse*, **29**: 183-192.
- Sharma, S.S. & Bhargava, H.N. (1998). Enhancement of morphine antinociception by ibogaine and noribogaine in morphine-tolerant mice. *Pharmacology*, **57**: 229-232.
- Shulgin, A.T. (1986). The background and chemistry of MDMA. *Journal of Psychoactive Drugs*, **18**(4): 291-304.
- Shulgin, A.T. & Shulgin, A. (1992). *PIHKAL [Phenethylamines I Have Known and Loved]: A chemical love story*. Berkeley, CA: Transform Press.
- Shulgin, A.T. & Shulgin, A. (1997). *TIHKAL [Tryptamines I Have Known and Loved]: The continuation*. Berkeley, CA: Transform Press.
- Snelders, S. (1998). The LSD therapy career of Jan Bastiaans, M.D. *Bulletin of the Multidisciplinary Association for Psychedelic Studies [MAPS]*, **8**(1): 18-20.
- Sprague, J.E., Huang, X., Kanthasamy, A. & Nichols, D.E. (1994). Attenuation of 3,4-methylenedioxymethamphetamine (MDMA) induced neurotoxicity with the serotonin precursors tryptophan and 5-hydroxytryptophan. *Life Sciences*, **55**(15): 1193-1198.

- Sprague, J.E., Everman, S.L. & Nichols, D.E. (1998). An integrated hypothesis for the serotonergic axonal loss induced by 3,4-methylenedioxymethamphetamine. *NeuroToxicology*, **19**(3): 427-442.
- Steele, T.D., McCann, U.D. & Ricaurte, G.A. (1994). 3,4-methylenedioxymethamphetamine (MDMA, “Ecstasy”): Pharmacology and toxicology in animals and humans. *Addiction*, **89**: 539-551.
- Stevens, J. (1987). *Storming heaven: LSD and the American dream*. New York: The Atlantic Monthly Press
- Stolaroff, M.J. (1997). *The secret chief: Conversations with a pioneer of the underground psychedelic therapy movement*. Charlotte, NC: Multidisciplinary Association for Psychedelic Studies (MAPS).
- Strassman, R.J. (1984). Adverse reactions to psychedelic drugs: A review of the literature. *Journal of Nervous and Mental Disease*, **172**(10): 577-595.
- Strassman, R.J. (1991). Human hallucinogenic drug research in the United States: A present-day case history and review of the process. *Journal of Psychoactive Drugs*, **23**(1): 29-38.
- Strassman, R.J. (1995). Hallucinogenic drugs in psychiatric research and treatment: Perspectives and prospects. *The Journal of Nervous and Mental Disease*, **183**(3): 127-138.
- Strassman, R.J. (1997). Biomedical research with psychedelics: Current models and future prospects. In R. Forte (Ed.), *Entheogens and the future of religion* (pp. 153-162). San Francisco, CA: Council on Spiritual Practices.
- Vollenweider, F.X., Gamma, A., Liechti, M. & Huber, T. (1999). Is a single dose of MDMA harmless? *Neuropsychopharmacology*, **21**(4): 598-600.
- Walsh, R. (1982). Psychedelics and psychological well-being. *Journal of Humanistic Psychology*, **22**(3): 22-32.
- Wasson, R.G., Kramrisch, S., Ott, J. & Ruck, C.A.P. (1986). *Persephone's quest: Enthegens and the origins of religion*. New Haven, CT: Yale University Press.
- Wei, D., Maisonneuve, I.M., Kuehne, M.E. & Glick, S.D. (1998). Acute iboga alkaloid effects on extracellular serotonin (5-HT) levels in nucleus accumbens and striatum in rats. *Brain Research*, **800**: 260-268.
- Weil, A.T. (1976). The love drug [MDA]. *Journal of Psychedelic Drugs*, **8**(4): 335-337.
- Yensen, R. (1985). LSD and psychotherapy. *Journal of Psychoactive Drugs*, **17**(4): 267-277.
- Yensen, R., Di Leo, F.B., Rhead, J.C., Richards, W.A., Soskin, R.A., Turek, B. & Kurland, A.A. (1976). MDA-assisted psychotherapy with neurotic outpatients: a pilot study. *The Journal of Nervous and Mental Disease*, **163**(4): 233-245.